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The tissue-specific self-pathogen is the protective self-antigen: the case of uveitis.

Mizrahi T, Hauben E, Schwartz M.

Department of Neurobiology, The Weizmann Institute of Science, 76100 Rehovot, Israel.

Vaccination with peptides derived from interphotoreceptor retinoid-binding protein (a self-Ag that can cause experimental autoimmune uveoretinitis) resulted in protection of retinal ganglion cells from glutamate-induced death or death as a consequence of optic nerve injury. In the case of glutamate insult, no such protection was obtained by vaccination with myelin Ags (self-Ags associated with an autoimmune disease in the brain and spinal cord that evokes a protective immune response against consequences of injury to myelinated axons). We suggest that protective autoimmunity is the body's defense mechanism against destructive self-compounds, and an autoimmune disease is the outcome of a failure to properly control such a response. Accordingly, the specific self-Ag (although not necessarily its particular epitopes) used by the body for protection against potentially harmful self-compounds (e.g., glutamate) can be inferred from the specificity of the autoimmune disease associated with the site at which the stress occurs (irrespectively of the type of stress) and is in need of help.

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